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Effects of Passive Smoking on Health of Children

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Analysis of data on the effects of passive smoking obtained in preadolescent children from the Harvard Six-Cities Study demonstrates an exposure-response relationship between the number of smokers in the household and the reporting rates for doctor-diagnosed respiratory illness before age 2, history of bronchitis, wheeze most days and nights apart from colds, and a composite of symptoms defined as the lower respiratory index. Similarly, when only the amount currently smoked by the mother was used, the data indicated a relatively uniform increase in each of the reported diseases and symptoms. FEV_1 was lower in children with smoking mothers compared to children of nonsmoking mothers. Rate of increases in FEV_1 after adjusting for normal growth was significantly smaller in children of smoking mothers and was related also to amount smoked. Notably the effect on level of FVC was not seen and this finding, consistent in several studies, remains unexplained.

Although children of smoking mothers were shorter on the average than children of nonsmoking mothers, no on-going passive smoking effect on height growth can be ascertained. All these differences are small and their medical significance remains to be defined.

Introduction

Recent studies of the health effects of air pollution have pointed out the importance of considering indoor pollutants as potential sources of additional, and potentially important pollution exposures (*1*). Sidestream cigarette smoke is one major source of indoor pollution. The impact of active cigarette smoking on respiratory health has been well documented (*2–4*). The effect of passive smoking is less clear. Some studies of passive smoking and respiratory health have shown an effect (*5,6*), while others have not (*7,8*). The reasons for these apparently inconsistent results are not entirely clear, but may arise from differences in sampling techniques, methods of analysis, study design and end points, small numbers of subjects and, in some cases, insensitive tests (*9*). These inconsistencies may also reflect real differences in indoor pollution associated with regional differences in ventilation of the homes.

The Six-Cities Study is a longitudinal study of children and adults designed primarily to assess the respiratory health effects of outdoor air pollution. Because of the potential confounding effect of passive smoking, as well as independent interest in its health effects, information on exposure to sidestream cigarette smoke has routinely been gathered at regular examinations of study participants. A number of analyses of these data have investigated the association between passive smoking and respiratory health of children. This paper summarizes the results of three such analyses (*10–12*). The results demonstrate an exposure-response relationship between the amount of passive cigarette smoking and most indices of respiratory health. There also appears to be an impact on height which may be related to *in utero* exposures. It remains to be determined whether these small effects have medical significance.

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Methods

Sample Selection and Data Collection

The six cities were selected to represent a range of air quality based on historically reported concentrations of SO_2 and particulate matter. The six cities were: Steubenville and Mingo Junction, OH; the southern tip of St. Louis, MO; Watertown, MA; Kingston-Harriman, TN; Topeka, KS; and Portage, WI and surrounding towns. The design of the study and selection of the communities have been previously described (*13*). The total cohort included 13,545 children between the ages 6 and 11 yr. Standard questionnaires on respiratory symptoms, home characteristics, and parental smoking and respiratory illness history were completed by a parent. Children in grade 4 or higher were privately asked about their personal cigarette smoking habits. Permission was obtained from the parents for the child to perform simple tests of pulmonary function which measured forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV_1) by means of a portable survey spirometer (Warren E. Collins Inc., Braintree, MA). Values were expressed at body temperature and pressure saturated (BTPS). Pulmonary function measurements were made with the child seated with free movement and without a nose clip. Standing height in stocking feet and weight were measured as well. Children were seen annually at school. This report describes analyses of data for white children, 6 to 9 years of age.

Cross-Sectional Analysis of Pulmonary Function, Respiratory Illnesses, and Respiratory Symptoms

From the questionnaires used at the first and second annual follow-up, the frequency of respiratory illness in the previous year which had kept the child at home 3 days or more, bronchitis in the previous year, the prevalence of persistent cough (for 3 months of the year or more) and persistent wheeze (most days or nights apart from colds) were analyzed. An index of lower respiratory illness was computed from these responses. This index included the presence of bronchitis, respiratory illness, or persistent cough during the previous year.

Pulmonary function values were transformed to their natural logarithms and then expressed as residuals or differences from height-, weight-, age-, and sex-specific values bases on our prediction equation from this population (*14*). These residuals from the initial and first follow-up visits were investigated for possible association with exposure to sidestream cigarette smoke. Results are reported as deviations from 100% of the ratio of observed to expected pulmonary function.

Parental smoking was categorized into three groups according to the reported smoking history (never smoked, ex-smoker or current smoker). Parental smoking was also coded by the number of cigarettes currently smoked per day by each parent. A positive history of parental respiratory illness was defined as a history of bronchitis, emphysema or asthma of either parent. To control for the association between socioeconomic status (SES) and respiratory health, analyses were adjusted for a three-level index of SES based on the mean number of years of schooling of the parents (<10, 10–12, >12 yr). In single-parent households the years of education of that parent were used.

Effect on Pulmonary Function Growth Rate

To assess the effects of passive smoking on development of pulmonary function, we estimated the pulmonary growth rate for each child having two or more annual visits and examined the association of growth rate with potential risk factors. Eighteen children who reported that they were smokers were not included in the analysis. The median number of visits per child was three.

Data were analyzed by an approximate growth curve method. From each child's series of pulmonary function residuals described above, we estimated the growth rate or slope by ordinary least squares regression of these residuals on age at the time of measurement. Estimated growth rates were then regressed on measures of exposure to cigarette smoke and potential confounding variables, including SES, city of residence, history of doctor-diagnosed respiratory illness before two years of age and bronchitis prior to initial examination. This analysis used weights determined by the within- and between-subject variation of regression coefficients (*15*).

Effect on Children's Height

This analysis was based on children who had at least two annual height measurements between the ages of 6 and 11 yr. The median number of visits per child was four. There were 224 children who reported they had ever smoked one cigarette a day or more, but their exclusion from the analysis did not affect the results.

Data were analyzed by the approximate growth-curve method similar to that used in the pulmonary function analysis. The height measurements of each child were expressed as deviations from age- and sex-specific predicted height (50th percentile) from the National Center for Health Statistics (NCHS) growth standards (*16*). Each child's new level was calculated by averaging these deviations for the period studied. The growth rate was calculated as the slope of a least-squares regression of the deviations on the ages at time of measurements. The level and growth rates were then regressed on measures of passive smoking and confounding factors such as exposure to gas cooking, city of residence and SES.

Results

During the first 7 yr of the Six-Cities Study, 13,545 children between 6 and 11 yr of age were seen at least once. There were 12,258 whites, 1,041 blacks, 90 Orientals, 126 American Indians or Mexicans, and 30 of other racial groups. We have so far limited our analysis of the effects of passive smoking to the white children. Of the 12,258 white children, there were 9838 (80.3%) with two or more examinations.

Cross-Sectional Analysis of Respiratory Symptoms and Pulmonary Function

We examined the reported respiratory symptoms and pulmonary function measurements of 10,106 white children 6 to 9 yr. of age at their first examination. We also examined the same data for the subset of these children seen one year later (N = 8380).

We have restricted our attention to two reported illnesses from the initial questionnaire: doctor-diagnosed respiratory illness before the age of 2 yr, and history of bronchitis, and two reported symptoms from the second examination—wheeze and an index of lower respiratory illness, i.e., any reported respiratory illness, bronchitis, or cough in the past year. Results for the complete set of respiratory illnesses and symptoms have been previously published (*10*).

For these reported respiratory illnesses and symptoms, all outcomes were found to be significantly associated with sex, age, and city of residence (Table 1). Controlling for these covariates by direct standardization for sex, age, and city, we also found a trend towards higher prevalence with lower socioeconomic status (SES) as measured by parental education, except for history of bronchitis which as a higher prevalence with higher SES (Table 1). The strongest potential confounder was a history of parental respiratory illness, which was associated with increases in prevalence of 72 to 155%.

The directly standardized rates also increased with the number of smoking parents. Reported rates were higher with one smoking parent than with none and with two compared to one (Table 2). Logistic regression, controlling for SES, was consistent with an additive effect of the number of smoking parents. Among those with only one smoking parent, rates were higher if the mother smoke rather than the father.

When we considered mother's smoking only, reported rates were higher for children whose mothers were current smokers compared to those whose mothers had never smoked. Rates were also elevated for the children of mothers who were ex-smokers (Table 2). These differences were all highly significant ($p < 0.01$).

To test the dose-response relationship among children whose mothers were current smokers, we estimated the odds ratio of respiratory illnesses and symptoms for various levels of maternal smoking, compared to the prevalence for children of mothers who had never smoked (Fig. 1). Children whose mothers were ex-smokers were excluded. A significant linear association ($p < 0.001$) was found for each outcome.

Other studies (*8*) have suggested that the association

Table 1. Frequency (rate per 1000) of reported illnesses and symptoms, by sex, age, city, parental education, and reported parental respiratory illness history directly standardized for sex, age, and city.

	1st examination		2nd examination	
	Doctor-diagnosed illness before 2 yr	History of bronchitis	Wheeze	Lower respir. index
N	9004	10608	8237	8240
Sex				
M	241	163	127	237
F	191	136	102	219
Age*				
6	227	155	—	—
7	212	148	121	239
8	222	151	113	228
9	183	153	120	213
10	—	—	92	194
City				
Portg	223	141	111	147
Topka	208	134	115	224
Wtrtn	129	113	89	216
Kngtn	341	195	148	313
StLou	198	127	111	204
Steub	221	210	125	281
Parent's education				
< High school	265	145	146	236
High school	220	150	118	225
> High school	195	157	94	228
Parent's illness				
No	174	116	91	184
Yes	299	261	184	330

*Excludes 44 seven-year-old and 9 eleven-year-old children at the second examination.

Table 2. Frequency (rate per 1000) of reported illnesses and symptoms, (and standard errors), by parental smoking directly standardized for age, sex, and city.

Smoking status	N at 1st exam	1st Examination		2nd Examination	
		Doctor-diagnosed illness before 2 yr	History of bronchitis	Wheeze	Lower respir. index
Mother					
Never	4044	193 (6)	137 (5)	99 (5)	205 (7)
Ex	1485	205 (11)	152 (9)	114 (9)	239 (12)
Current	4208	212 (7)	159 (6)	128 (6)	243 (7)
Unknown	372				
Father					
Never	1832	185 (10)	135 (8)	104 (8)	202 (10)
Ex	1784	204 (10)	160 (9)	106 (8)	222 (10)
Current	5171	229 (6)	149 (5)	119 (5)	231 (7)
Unknown	1319				
Current parental					
Neither	2726	173 (12)*	130 (10)*	99 (6)	206 (8)
Father only	2193	203 (8)	141 (7)	107 (7)	217 (9)
Mother only	817	209 (17)	151 (15)	117 (12)	227 (16)
Both	2792	235 (7)	160 (6)	131 (7)	240 (9)
Unknown	1578				

*Based on smoking history rather than current smoking.

Table 3. Odds ratios for respiratory illnesses and symptoms by reported presence of respiratory illness in parents and mother smoking 1 pack/day, with and without adjustment for parental respiratory illness history; all are controlled for age, sex, and city-cohort.

	N	Parent's illness only	Mother's smoking only	Mother's smoking controlled for parent's illness
Doctor-diagnosed respiratory illness before age 2	6676	2.05‡	1.35‡	1.28‡
History of bronchitis	6675	2.77‡	1.16†	1.07
Wheeze	5385	2.30‡	1.31‡	1.23†
Lower respiratory index	5390	2.32‡	1.23‡	1.16*

*$p < 0.01$.
†$p < 0.001$.
‡$p < 0.00001$.

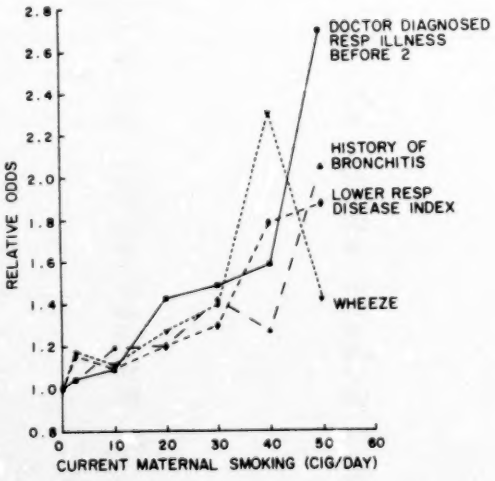

FIGURE 1. Relative odds of respiratory illness or symptoms vs. average daily cigarette smoking by the child's mother. Reference value is zero cigarettes per day.

of increased symptoms with passive smoking disappears when the analysis is controlled for increased reporting of children's respiratory illness by parents with respiratory illnesses. As noted earlier, we found a strong association between children's illnesses and a history of respiratory illness in the parents. We estimated the effect of parental illness history and maternal smoking of one pack per day separately in a logistic regression (Table 3). Controlling for parental respiratory illness history reduced the estimated effect of maternal smoking, but a positive relationship remained. The association is, in fact, still statistically significant for three of the four outcomes. Furthermore, controlling for parental illness history may represent overadjustment, since the effect of controlling for parental illness history may also partly control for parental smoking—the potential proximal cause of the parents' symptoms.

FEV_1 adjusted for height, weight, and age also had a significant ($p < 0.001$) linear relation with the mother's reported current smoking, at both the initial and follow-up examinations (Fig. 2). The estimated effect of exposure to one pack per day was small, at both the initial

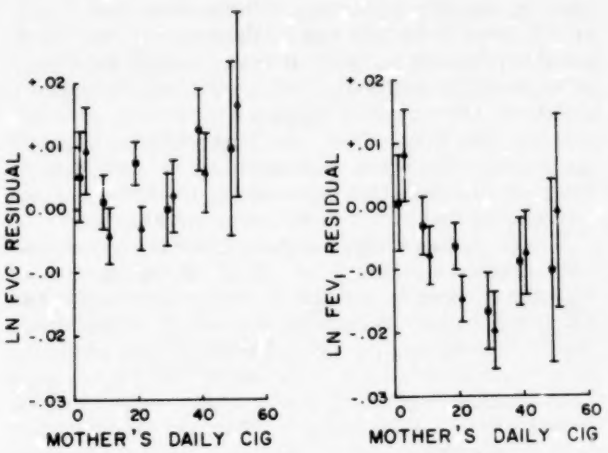

FIGURE 2. Mean logarithm of pulmonary function residuals (± one standard error) by mother's reported daily cigarette smoking, compared to children whose mothers have never smoked: (■) first examination (N = 7112); (▲) second examination (N = 6278).

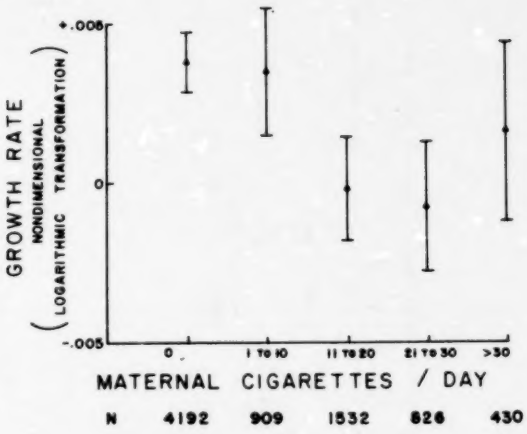

FIGURE 3. Mean deviation of logarithmic growth rate from average growth vs. mean number of cigarettes smoked daily by mother.

and first follow-up examinations (–0.7 ± 0.2% and –0.8 ± 0.2%, respectively). For FVC a positive association was noted at the initial examination (+ 0.5% ± 0.2%) and similarly, although not significantly, at the follow-up examination (+0.2 ± 0.2%). We expected that level of pulmonary function should reflect the cumulative lifetime exposure of the child to cigarette smoke and other environmental insults. Reported current maternal smoking may not be a good indicator of that cumulative exposure.

Effects of Passive Smoking on Pulmonary Function Growth Rate

After subtracting the predicted FEV_1 value based on the normative model (*14*) for each measurement, the mean growth rate among children with three or more examinations was not significantly different from zero (*12*). Regression analysis indicated that respiratory illness before age two, history of bronchitis, exposure to gas cooking fuel and smoking by the child's father were not predictors of plumonary function growth rate. Figure 3 illustrates how the estimated pulmonary function growth rate tends to decease as exposure to maternal smoking increases. When controlling for city of residence and SES, only maternal smoking demonstrated an association with growth rate ($p < 0.02$), A preadolescent child having a mother who smoked one pack per day over the period of a year was estimated to grow 0.22% of predicted FEV_1 less than the child of a nonsmoker. This means that if a group of children having identical FEV_1's at age 6 are followed to age 10, and the mothers of one group smoke a pack of cigarettes daily, then by age 10 the children of the smokers will have FEV_1 that are on average 15.6 mL smaller than the FEV_1 of the children of nonsmokers.

This finding (*12*), in conjunction with the cross-sectional results, suggests that children of smoking mothers have reduced FEV_1 and experience continuing loss of pulmonary function relative to population norms when passively exposed to cigarette smoke.

Effects of Passive Smoking on Attained Height and Growth

There was a clear and significant (p = 0.0001) trend in the attained height level of children grouped by the number of cigarettes smoked by the mother: 0, 1–9, and 10+ cigarettes daily (Fig. 4). This association was significant when tested in a multiple regression model adjusting for potentially confounding factors (*11*). Children of light smokers were on the average 0.45 cm shorter than children of nonsmokers, while children of heavy smokers were 0.65 cm shorter. Paternal smoking also showed a negative association with child's height (when tested along with maternal smoking), but it was not statistically significant.

Rate of growth in height for these children was not significantly associated with either maternal or paternal cigarette consumption (*11*). This suggests that, unlike pulmonary function growth, height growth during preadolescence is negligibly associated with continued passive exposure to cigarette smoke. Therefore the observed deficit most likely occurs as a result of *in utero* or early-life exposure to products of maternal smoking, and is essentially over before age 6 yr.

Discussion

The findings presented represent a relatively brief overview of the data we are collecting in the Six-Cities Study that relate to the issues of the effects of passive smoking on growing children. The consistency of the results is not in itself sufficient to prove an effect of

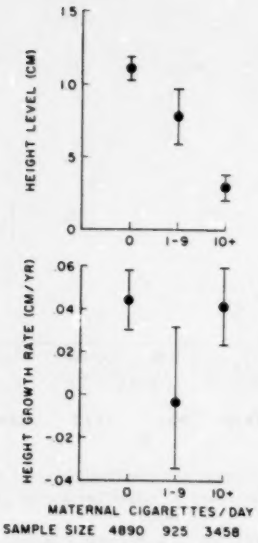

FIGURE 4. Mean deviation of height level and growth rate (± standard error) from average value for child's age and sex vs. mean number of cigarettes smoked daily by mother.

passive smoking. The results are, however, compelling and need to be considered seriously.

The biologic basis of our understanding of lung injury from respiratory irritants includes basic mechanistic studies of particle deposition and clearance (*17*). Extracts of tobacco smoke can delay ciliary action and are associated with increased infectivity in animal studies (*18*). Clinical studies of both patients and normal subjects have demonstrated pathologic changes in airways that are both indirectly and directly attributable to cigarette smoking (*19*). Almost every well conducted population-based study which has inquired into the effect of cigarette smoking has found an adverse health effect from smoking.

Investigations of passive exposure suggest that exposure is related to both the number of smokers and the time (frequency of smoking) and the ventilation rate of the space in which the smoking is occurring (*20*). In households, the level of respirable particles is related to the background outdoor level plus approximately 20 to 30 $\mu g/m^3$ for each smoker living in the household (*21*). Since the children in the study spend approximately 60 to 80% of their time inside their homes and 68% of the children live in a household with a smoker (*10*), exposure is likely for a majority of children.

Most of the studies that indicate a passive smoking effect find a better correlation with maternal smoking than with paternal smoking. This is consistent with the fact that mothers of young children tend to spend more time at home with their children than do fathers, and thus, if these mothers smoke, they are more likely to be smoking in the home with their children present.

At present, insufficient data exist to determine just when the effect on lung growth occurs. Too few mothers, who were smokers during their pregnancies and gave up smoking when their children were less than 5 yr old, have been followed to determine if the effect noted is primarily an *in utero* or early childhood effect, or an on-going continuous exposure effect throughout childhood. Our own data suggest a persistent, although possibly lessening, effect. As these children begin to move outside the home, their exposure at home lessens, however, and their own personal exposure may become a secondary and in fact more important risk factor.

Not all studies of children have found the association with passive smoking (*7,8*). Most of the studies of younger children that failed to find an association had either quite small populations studied (*7*) or used techniques of adjusting for other familial effects that may have "over matched" for the smoking effect (*8*) or have been studies in children from climatic evirons which were relatively mild (*8*) and in which the children were from homes with higher ventilation rates than were the children in our cities.

The effect of cigarette smoking of the mother on the attained height in children has been previously recognized. The fact that we were unable to demonstrate an ongoing effect on rate of growth in height suggests the effect to be an *in utero* or at least an early-life effect. Infarcts on the palcenta have been demonstrated on placentas obtained from smoking mothers, and the suggestion has been made that either reduced nutrient absorption and/or increased carboxyhemoglobin or nicotine levels may contribute to the smaller birth weight (*22*).

Finally, in considering the importance of these findings in the context of the development of chronic obstructive pulmonary disease (COPD) in later life, it is unlikely that even lifelong exposure to passive cigarette smoking alone will lead to a significant portion of the population developing COPD. However, even modest reductions in maximum obtained pulmonary function in childhood and early adult life have been suggested to be a risk factor for the development of more serious disease upon adult exposure to other putative agents (*23*). The proof of this hypothesis will require completion of other on-going investigations. Meanwhile, we shall persist in trying to characterize the effect of passive exposure to cigarette smoke in our sample of children, as well as investigating how passive smoking relates to the likely possibility that 20 to 25% of them will take up active smoking in the next few years.

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